## **Amendments to the Claims:**

This listing of claims will replace all prior versions, and listings, of claims in the application:

## **Listing of Claims:**

Claim 1 (previously presented): A process for identifying inhibitors of a human potassium channel,

- a) providing a mutated *S. cerevisiae* cell which does not express the three endogenous potassium channels TRK1, TRK2 and TOK1 and which is not complemented by an expressed HERG1;
- b) treating said mutant with a human potassium channel aside from HERG1 wherein said human potassium channel is expressed heterologously in this mutated *S. cerevisiae* cell;
- c) incubating the *S. cerevisiae* cell expressing the human potassium channel together with a substance to be tested; and
- d) determining the effect of the substance to be tested on the human potassium channel, wherein a decrease in the transport of potassium across the human potassium channel indicates that the substance is an inhibitor of the human potassium channel.

Claim 2 (previously presented): The process as claimed in claim 1, wherein the genes TRK1, TRK2 and TOK1 are switched off in the mutated *S. cerevisiae* cell ( $\Delta$ trk1,  $\Delta$ trk2,  $\Delta$ tok1).

Claim 3 (cancelled)

Claim 4 (currently amended): The process as claimed in claim 1, wherein the human potassium channel is Kv1.5 or gpIRK1.

Claim 5 (previously presented): The process as claimed in claim 4, wherein the human potassium channel is mutated.

Claim 6 (previously presented): The process as claimed in claim 2, wherein the human potassium channel is present in a yeast expression plasmid.

Claim 7 (previously presented): The process as claimed in claim 2, wherein the mutated *S. cerevisiae* cell expresses constitutively a growth reporter.

Claim 8 (previously presented): The process as claimed in claim 7, wherein the substance to be tested, which has an effect on the human potassium channel, inhibits the growth of the mutated *S. cerevisiae* cell.

Claim 9 (previously presented): The process as claimed in claim 7, wherein the effect of the substance to be tested on the human potassium channel is determined by measuring the cell count of the mutated *S. cerevisiae* cells.

Claim 10 (previously presented): The process as claimed in claim 9, wherein the cell count is determined via the fluorescence or luminescence of the constitutively expressed growth reporter.

Claims 11-19 (cancelled)

Claim 20 (previously presented): A process of identifying activators of a human potassium channel,

- a) providing a mutated *S. cerevisiae* cell which does not express the three endogenous potassium channels TRK1, TRK2 and TOK1 and which is not complemented by an expressed HERG1;
- b) reacting said mutant with a human potassium channel aside from HERG1 wherein said human potassium channel is expressed heterologously in this mutated *S. cerevisiae* cell;
- c) incubating the *S. cerevisiae* cell expressing the human potassium channel together with a substance to be tested; and
- d) determining the effect of the substance to be tested on the human potassium channel wherein an increase in the transport of potassium across the human potassium channel indicates that the substance is an activator of the human potassium channel.

Claim 21 (previously presented): A process of identifying activators of a human potassium channel,

- a) providing a mutated *S. cerevisiae* cell which does not express the three endogenous potassium channels TRK1, TRK2 and TOK1 and which is not complemented by an expressed HERG1;
- b) reacting said mutant with a human potassium channel aside from HERG1 wherein said human potassium channel is expressed heterologously in this mutated *S. cerevisiae* cell;

- c) incubating the mutated *S. cerevisiae* cell expressing the human potassium channel together with a substance to be tested in the presence of an inhibitor of the human potassium channel; and
- d) determining the effect of the substance to be tested on the human potassium channel wherein an increase in the transport of potassium across the human potassium channel indicates that the substance is an activator of the human potassium channel.

Claims 22-25 (canceled)